

PHILADELPHIA NEUROLOGICAL SOCIETY.

February 28 1905.

The President, Dr. Joseph Sailer, in the Chair.

A Case of Lumbo-thoracic Syringomyelia.—This case was exhibited by Dr. Ralph Pemberton for Dr. William G. Spiller. The patient, an Italian woman, aged 36 years, said she had always been healthy as a child, except for some eruptive fever affecting the face. She had never been to a hospital until admitted for the present complaint. She was married ten years ago and had a child eight years ago, which lived three days. She has had no miscarriage, and though she has been in America for eight years and in the hospital for the past two years, she cannot speak English.

The history of her present illness is very vague, and little can be learned beyond the fact that five years ago weakness in her legs, difficulty in walking and pain in her back developed. Her intelligence and her dialect are too poor to furnish further dependable data, even through an interpreter.

The pupils are slightly unequal, the right being larger than the left, but they both react promptly to light and in accommodation and convergence. The vertebral column shows no deformity.

The motion, coördination and sensation of the arms and hands are good. The reflexes are about normal in intensity, the left biceps jerk being, perhaps, slightly increased.

The legs are well developed and show no trophic disturbance in any part. In the right leg and thigh voluntary power is about normal, but in the left leg and thigh it is distinctly diminished, though not lost. On the left side are also a greatly increased patellar reflex, distinct patellar clonus, persistent ankle clonus and a typical Babinski sign. On the right side the patellar reflex and the Achilles jerk are completely lost, but Babinski's sign is distinctly present, though not quite so pronounced as upon the left. Over both legs light touch is promptly perceived, but on the right side pain sensation is undoubtedly diminished, though pin prick is felt if the prick be deep. Over the whole right lower limb sensation for heat is much diminished, and also apparently over the lower part of the abdomen on the right side, but over the left leg and thigh perception of heat and pain is very prompt. Sensation of cold probably is also diminished in the entire right lower limb.

The gait of the patient is peculiar, as owing to the spasticity of the left leg, she swings the leg somewhat from the hip and scrapes the toes of the left foot along the ground. The right lower limb is thrown somewhat unnecessarily forward in walking and the heel is brought down before the ball of the foot, and the foot describes a small arc outward.

Some spasticity develops on passive motion of the left leg, but the right leg is entirely flaccid and the heel-to-knee movement, which cannot be performed on the left side on account of weakness, is easily and co-ordinately performed upon the right side. There is no ataxia of station.

The general health is good. She has no disturbance of bladder or rectal functions, though two years ago she developed incontinence of urine for a short time. Her temperature, pulse and respiration are normal.

The Babinski Sign Occurring in Strychnine Poisoning and Acute Lead Encephalopathy.—This report was made by Dr. Ralph Pemberton.

Case I.—A white female, age 30 years, with diagnosis of melancholia and possibly petit mal, took between $1\frac{1}{2}$ and 2 grains of strychnine sul-

phate. Pain and rigidity in the feet and legs developed in about ten minutes, with intermittent convulsions, which grew more frequent, and finally became general. Bilateral ankle clonus was present at the end of the first ten minutes of the attack, with a plain and prompt Babinski sign on the right side. Urgent need of treatment prevented testing for it on the left side. Babinski's sign and ankle clonus had not been present before the attack, and were not present after an uneventful recovery, though the patellar reflexes were somewhat increased.

Case II.—A colored male, aged 23 years, presented a history of working in lead for nine months, and in that time had had two attacks of malaise, abdominal pains, nausea and vomiting. The second attack was accompanied by unconsciousness.

The patient was seen about 34 hours after the onset of a third attack, which was similar to the second except that the third was more severe. He was then in pronounced stupor, could be roused with great difficulty to mutter inarticulately, and presented a plain Babinski sign on the left and a doubtful one on the right side. The reflexes elsewhere in the legs were depressed. On the following day Babinski's sign was still present, and could also be elicited on the left side by stroking the sole of the right foot; *i. e.*, it was a crossed Babinski phenomenon. On the next day the Babinski sign had entirely disappeared. The patient was by this time almost clear mentally, and made an uneventful recovery. The diagnosis was lead encephalopathy.

(Case I) *Progressive Muscular Dystrophy, with Atrophy of Bone*; (Case II) *Chronic Mercurial Poisoning*, and (Case III) *Tabetic Facial Palsy*.—These cases were exhibited by Dr. William G. Spiller.

Case I.—Muscular dystrophy had existed since the second year of life. The man had had no acute disease and never any pain. Sensation was not affected. He had marked atrophy of all parts of the body, and the atrophy had progressed gradually and had become intense, and was asymmetrical in some parts. Dr. Spiller thought this was not a case of poliomyelitis because of the history. The humerus on the left side was markedly smaller than that on the right side.

Case II showed a rare condition of chronic mercurial poisoning. There was marked tremor, increased by active movement. At times this tremor was present in all parts of the body, even in the soft palate and larynx. The larynx was congested, suggesting disturbance of the liver. Degenerative changes were seen in the optic discs. The speech was tremulous.

Case III.—This was one of tabes in which the diagnosis was made because of loss of the patellar tendon reflexes, loss of the Achilles tendon reflexes, occasional shooting pains in the legs, some difficulty in holding the urine, ptosis of the left upper eyelid, paresis of the left external rectus muscle, paralysis of the right external rectus muscle, irregularity of the pupils, Argyll-Robertson pupils and optic atrophy. In addition the man had paresis of the right side of the face, of which he was entirely ignorant.

Dr. Dercum thought Case I was remarkably interesting, inasmuch as the man had changes not only in the limbs, but in the bones of the face, the right malar bone being smaller and the zygomatic arch narrower than the corresponding structures on the left side. He thought there was something in the face to suggest hemifacial atrophy, while the limbs called to mind some of the appearances of morphea. No one has ever explained the relation between morphea and these deep-seated dystrophies. That there is some relation is extremely probable. We are entirely in the dark with regard to their pathogenesis. The only autopsy of a facial hemiatrophy which Dr. Dercum could recall was that of Mendel, made many years ago. Mendel found a degeneration of the descending root of the fifth nerve. In the present case there are no skin changes such as we are apt to find in hemifacial atrophy. Dr. Der-

cum thought the case went far to prove the kinship between atrophic and dystrophic diseases.

Dr. Lloyd stated that this case recalled to his mind a case he reported to this Society some years ago of pseudo-hypertrophic muscular paralysis with trophic changes in the joints. That case was of the pseudo-hypertrophic type and not the atrophic type. The changes were principally in the elbows and knees.

Dr. Mills referred to a case he had reported several years ago where the symptoms were very similar to those of Dr. Spiller's Case II. The patient had worked in the same factory where Dr. Spiller's patient was employed. His case had some of the features of general paralysis, notably tremor, peculiar tremulous speech and a depressed mental condition.

Dr. Dercum thought it very remarkable that when the poisoning by mercury takes place through the respiratory tract the symptoms are so different from those present in poisoning caused by hypodermic administration, by inunction or by internal administration. The question arises whether we have not a mixed intoxication to deal with, *i. e.*, one due not solely to mercury. It is interesting, too, to note that many of the symptoms resemble those of chronic alcoholic poisoning.

In connection with Dr. Spiller's third case (tabetic facial palsy) Dr. Gordon mentioned a case he saw several years ago of tabes in which facial palsy occurred six months after infection. The symptoms of tabes made their appearance one year after the infection. The facial palsy was not a paresis, but a true Bell's palsy. After the patient was cured of palsy the tabetic symptoms became much aggravated.

Dr. Spiller, in closing the discussion on his cases, stated that in 1898 he had reported a case of muscular dystrophy with involvement of bone. In regard to the man with chronic mercurial poisoning, he said the gums were much affected.

Hemorrhage in the Floor of the Aqueduct of Sylvius.—Dr. Weisenburg exhibited a patient with hemorrhage into the nuclei of the third and fourth nerves. This patient was seen with Dr. M. Radcliffe in the Wills Eye Hospital. The man, 53 years of age, a farmer, while pitching hay suddenly felt a "gripping feeling" in both eyes which was followed almost immediately by ptosis of both eyelids. This has persisted. He had never been seriously sick previously and had no venereal history. A slight frontal headache with some vertigo was present occasionally both before and since the attack, but no other symptoms were complained of.

With the face at rest there was complete ptosis of both eyelids, it being possible to see the cornea for about 1-16 of an inch. When he was looking upwards forcibly the cornea could be seen for 3-16 of an inch in the right eye and 2-16 in the left. The eyelids could be shut forcibly and well on each side. Dr. Radcliffe stated that vision in O. D. was 20-30, in O. S. 20-40. The right pupil was oval and 4.5 mm. in width, the left round and 4.5 mm. in width. No reaction to light, accommodation and convergence could be obtained. There was complete inability to move the eyeballs upward, downward or inward; external rotation being possible on each side, but weak. The ophthalmoscopic examination showed the discs to be pale, but the fundi were otherwise normal.

All of the other cranial nerves were normal. Hearing and taste were not affected. All the limbs were normal in motion, the reflexes were not altered, and sensation was not disturbed.

The sudden onset without any previous symptoms of any kind suggested most probably a diagnosis of hemorrhage in the nuclei of the third and fourth nerves.

Hemorrhage in the Pons.—Dr. Weisenburg also exhibited a patient who had a fracture of the base of the skull, which probably caused a

hemorrhage in the right side of the pons. This patient was studied in the Polyclinic Hospital, in the service of Dr. Spiller. Two years ago this man, who is 48 years old, fell from the back of a trolley car and was said to have struck the back of his head. He was taken to a hospital in an unconscious condition and remained so for four days. A diagnosis of a fracture of the base of the skull was made. When he regained consciousness he was found to be paretic on the left side of the body, and he saw double. He was able to go home three weeks after the accident, at which time the paresis of his left side had almost completely disappeared. He has complained ever since of a feeling of numbness in the left forearm and of a "chilly" feeling in his left leg. He has never had headache, nausea, vomiting or convulsions, but his memory has become poor. The diplopia has persisted.

Examination showed the pupils to be of equal size. The response to light, accommodation and convergence was normal. The right external rectus was paretic. The eyegrounds were normal. The right seventh nerve was weak, but this could be attributed, possibly, to the paralysis of the sensory part of the fifth nerve, and was a sensory paralysis. The left seventh nerve was normal. The man complained of a constant roaring in his right ear, though hearing was normal. The masseter on the right side did not contract as well as it should, and a constant fine fibrillary tremor in the temporal and masseter muscles was present. There was a complete anesthesia for touch and pain in the upper distribution of the right fifth nerve and a hyperesthesia in the middle and lower distribution. There was no keratitis neuroparalytica. The patient complained of a constant feeling of heaviness in the right side of his face, this being due to involvement of the fifth nerve.

The tongue, when protruded, deviated to the right, and constant fine fibrillary tremors were present in both sides, more marked on the right. There was also a suggestion of atrophy on either side of the tongue. The sense of smell was preserved. Lachrymation was present on irritation of the left nostril, and not on the right side. Both the upper and lower limbs were normal in power and movement. Sensation was preserved in all parts of the body. All of the tendon reflexes were exaggerated and unequally so. There was no Babinski reflex and no ataxia. The bladder and rectal functions were normal.

A hemorrhage in the right side of the pons would best explain the symptoms in this case, for a lesion in this area would involve the fifth, sixth, seventh and possibly the eighth nerves, all of which were implicated in this case. The irritative sensory phenomena present in the left side of the body were probably caused by a lesion of the median fillet, a so-called central pain. Such cases are rare, but are to be found in the literature. The early hemiplegia was merely a transient symptom, and due, no doubt, to involvement of the pyramidal fibers. The bilateral involvement of the hypoglossus nerve can be explained by an extension of the hemorrhage into the nuclei of these nerves.

Dr. Dercum asked Dr. Weisenburg why he did not call the first case one of polioencephalitis superior. The symptoms of this disease may come on very quickly. Why call it hemorrhage?

Dr. McCarthy stated that he had the same thought as Dr. Dercum about this case. If the entire third nerve were involved on both sides it would necessitate a rather extensive and bilateral hemorrhage, and he thought it would be more consistent to make a diagnosis of polioencephalitis superior.

Dr. Spiller said that the symptoms of polioencephalitis superior might come on within a short time, but he thought it would be difficult to say in regard to this case whether the symptoms were due to polioencephalitis superior or to hemorrhage. He thought, however, that the distinction

was not very important, as in cases of polioencephalitis often there are small hemorrhages, and a lesion need not be very great to destroy the oculomotor nucleus. It would have to be a bilateral hemorrhage, of course, but he had seen hemorrhage in the tegmentum of the pons go beyond the raphe and extend to the other side.

Dr. McCarthy stated that he had specimens of polioencephalitis superior which showed miliary hemorrhages and marked degeneration of the nuclear cells. The miliary points of hemorrhage were like what is seen in the anterior horn cells in anterior poliomyelitis.

Dr. Weisenburg, in closing the discussion on his case of hemorrhage in the floor of the aqueduct Sylvius, stated that it would be rather difficult to make a diagnosis between superior polioencephalitis of Wernicke and hemorrhage, but in a man who had had no symptoms at all and in a moment bilateral ptosis developed one would naturally look for hemorrhage, rather than polioencephalitis.

A Case of Malarial Infection Presenting Symptoms of Multiple Neuritis.—This report was read by Dr. George E. Price.

Dr. Weisenburg stated that while in the Philippines he saw a great deal of malaria, and a great many nervous symptoms were present in those cases. He mentioned the case of a surgeon who became suddenly weak in both knees and fell. He was taken to the hospital and found to have all the symptoms of multiple neuritis, due to malaria. He stated that he had seen perhaps six cases of malarial neuritis in the two years he was there. So far as treatment was concerned, quinine was given in massive doses. Occasionally a vein was opened and 50 or 60 grains injected.

Clinical and Pathological Report of a Case of Lead Poisoning, with Remarks on the Pathogenesis of the Disease.—This paper was read by Dr. Alfred Gordon.

Dr. Dercum thought the interesting point in this report was the posterior sclerosis. Posterior sclerosis as a result of chronic lead poisoning is a novel finding. It is most suggestive as to the action of poisons in general.